

Rolles, C. J., Kendall, M. J., Nutter, S., and Anderson, C. M. (1973). One-hour blood-xylose screening test for coeliac disease in infants and young children. *Lancet*, 2, 1043.

Sammons, H. G., Morgan, D. B., Frazer, A. C., Montgomery, R. D., Philip, W. M., and Phillips, M. J. (1967). Modification in the xylose absorption test as an index of intestinal function. *Gut*, 8, 348.

We showed the letter by Dr. Rolles and his colleagues to Dr. Lamabadusuriya and his colleagues, who replied as follows:

The purpose of our article was to draw attention to the limitations of the xylose tolerance test as a screening procedure for coeliac disease in childhood, and we note that Dr. Rolles and colleagues agree with us that there are no fully satisfactory screening tests available at present. In the majority of children the diagnosis of coeliac disease is made before 5 years, an age when accurate timed collections of urine are notoriously difficult to obtain, even in a well-staffed ward. On the basis of our results for the 5- and 24-hour urinary xylose outputs we are of the opinion that the urinary xylose test should be abandoned in the paediatric population. We did not assess the value of the 2-hour urine xylose test, since we strongly suspected that this would also be indiscriminate. The loading dose of xylose in our study was determined according to body weight (0.4 g/kg up to a maximum of 7.5 g/kg) and was administered as an isomolar solution (3%), for the reasons discussed in our paper. Variation in total dose of administered xylose is an unlikely explanation for our finding that 2 children with newly diagnosed coeliac disease had normal 1-hour blood xylose levels, since they received 4 and 4.8 g of xylose as a loading dose.

The comments by Rolles and colleagues on the relation between xylose transport mechanisms, total dose of xylose, and 1-hour blood xylose levels may be a misinterpretation of the kinetics of xylose transport. Initial luminal disappearance rates of xylose from the proximal small intestine (and therefore 1-hour blood levels) are likely to be related more closely to the luminal concentrations of xylose rather than to the total dose administered, whether transport across the mucosa is by a process of passive diffusion or by an active carrier-mediated one. In our study all administered solutions contained the same concentration of xylose (3%) and, assuming an approximately similar degree of dilution of the oral load, xylose concentrations in the proximal small intestine would be anticipated to be similar; thus it is unlikely that the total dose of xylose would appreciably influence the 1-hour blood level.

Penicillin has been shown to inhibit D-xylose transport in rat intestine (Giorgi, 1970) and presumably would do so in the human, since the transporting mechanism is the same in both species.

It is now well established, of course, that the histological appearances of the jejunal mucosa in coeliac disease are not specific; specificity lies only in the temporal relation between dietary gluten withdrawal or reintroduction, and the associated mucosal changes. An unequivocal diagnosis of coeliac disease can only be made when such a specific relation has been shown by means

of sequential biopsies and a carefully controlled dietary intake of gluten.

We consider that Rolles and colleagues (1973) somewhat overstated their case for the 1-hour blood xylose test, e.g. '... a normal one-hour blood xylose test almost certainly excludes coeliac disease'. We should like to make the simple plea that a normal 1-hour blood xylose does not exclude coeliac disease.

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Diabetes mellitus after mumps vaccination

Sir,

There are many published reports indicating a temporal relationship between antecedent mumps infection and the development of diabetes mellitus (Harris, 1899; Patrick, 1924; Kremer, 1947; Hinden, 1962; McCrae, 1963; Messaritakis, *et al.*, 1971). Overt diabetes appeared in all reported cases within weeks after a natural infection with mumps virus. No case of diabetes mellitus has been reported after infection with an attenuated mumps virus as in the present case.

A 6½-year-old boy was admitted in May 1973 with a history of polydipsia, polyuria, and bed-wetting of 5 days' duration. One month before onset of symptoms the patient had received mumps vaccine (Mumpsvax). Physical examination on admission was unremarkable. Urine examination showed glycosuria (1 g/dl) and 1+ reaction for ketones. Blood sugar was 345 mg/dl. Serum and urine amylase were normal. He was soon stabilized on 5 units of lente insulin. He has been followed regularly and his diabetes persists 30 months later.

Family history was negative for diabetes, and in both parents an oral glucose tolerance test was normal.

The role of mumps infections in the development of diabetes mellitus was suggested a long time ago (Harris, 1899), and it has been the subject of considerable controversy. Because of well-known association of mumps with pancreatitis, it has been suggested that diabetes mellitus, following mumps infection, is the result of damage to the islet cells though in the majority of reported cases, as in ours, clinical or laboratory evidence of pancreatitis was lacking. Another possibility is that in our patient a latent state of diabetes was activated by the attenuated mumps virus contained in the vaccine.

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Hypernatraemic dehydration and infant mortality

Sir,

Discussion of the most appropriate treatment of hypernatraemic dehydration in infancy (Bannister, Matin-Siddiqi, and Hatcher, 1975), together with reports on the connexion between that condition and sudden death in infancy (Emery, Swift, and Worthy, 1974), has led us to report our experience at the Royal Liverpool Children's Hospital in 1972 and 1973.

During this period 4 of 14 children admitted with hypernatraemic dehydration died, a mortality of 28%. All 4 deaths were in infants under 6 months of age. 3 of the babies who died (Cases 1, 2, and 3) were admitted *in extremis* with circulatory failure and severe electrolyte

and acid-base disturbance and with a very brief history of illness (see Table). It can be speculated therefore as to how much longer these infants who did not show an effective response to resuscitation, could have remained at home before dying 'unexpectedly'.

Not only is hypernatraemic dehydration a significant cause of sudden death in infancy, but the point must also be made that as much can be done to prevent as to treat the condition. There was evidence that 11 of the 14 cases, including all 4 infants who died, consistently received feeds containing excessive solute and calories. None of the 14 had been breast fed.

We therefore add our plea to that of other contributors to your journal for more ready recognition of those factors in infant nutrition, particularly hyperosmolar feeding, which lead to hypernatraemic dehydration and thus contribute appreciably to mortality in early childhood.

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TABLE

Case no.	Sex	Age (w)	Duration of illness before admission (h)	Initial serum sodium (mEq/l)	Initial blood urea (mg/dl)	Initial base deficit (mEq/l)
1	F	5	24-36	184	200	15
2	F	6	12	169	189	Not done
3	F	12	<6	153	57	20
4	M	22	120	155	110	6